

CASE REPORT

Hemodynamic Uncoupling and Sympathetic Surge Attenuation in a Geriatric Patient with Cardiometabolic Multimorbidity During Open Cholecystectomy: A Case Report

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ABSTRACT

Background. Older surgical patients with cardiometabolic multimorbidity have little physiological reserve. Stiff arteries and diabetic autonomic neuropathy can separate heart rate from blood pressure during surgical stimulation and hide an afterload surge.

Objective. To describe hemodynamic uncoupling and a risk-adapted anesthetic plan in an older patient undergoing open cholecystectomy.

Methods. A 71-year-old man (ASA III, Clinical Frailty Scale 3) with hypertension, coronary disease, insulin-treated type 2 diabetes and left ventricular hypertrophy had an open cholecystectomy under general anesthesia. Blood pressure was measured by oscillometry every 1 to 2 minutes with continuous ECG ST-segment analysis. Fluids were restricted, extubation was deep against five criteria, and a low-dose fentanyl infusion with ward safety measures followed.

Results. Traction on the gallbladder mesentery raised blood pressure from 138/84 to 171/95 mmHg (MAP 120.3 mmHg) while heart rate rose only from 72 to 83 bpm. After sevoflurane was increased to 3.5% and fentanyl 50 mcg was given, systolic pressure was 148 mmHg 4 minutes after the surge began, and no ST-segment depression was recorded. Creatinine stayed stable (1.10 to 1.00 mg/dL) after 500 mL of crystalloid. Extubation was uneventful, resting pain stayed at or below 2/10, and he went home on postoperative day 3 without cardiovascular, pulmonary or renal events.

Conclusion. A rise in systolic pressure without tachycardia was the only sign of a visceral nociceptive surge. One-minute non-invasive pressure measurement, deeper volatile anesthesia and a small opioid bolus controlled it within minutes. One case cannot show that invasive monitoring is unnecessary.

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1. Introduction

Older surgical patients have limited physiological reserve (homeostenosis), and cardiometabolic multimorbidity (hypertension, coronary artery disease and type 2 diabetes mellitus) reduces it further.^{1–6} Frailty is common in this group.^{4,7} In diabetes, chronic hyperglycemia leads to advanced glycation end-products that cross-link collagen in the arterial media, and the aorta loses its Windkessel compliance.^{8–10} A brief rise in stroke volume or catecholamines then produces a steep rise in systolic pressure, which raises left ventricular wall stress and myocardial oxygen demand by the law of Laplace.

Diabetic cardiovascular autonomic neuropathy adds a second problem. It is associated with arterial stiffness,^{11–17} and it blunts the baroreflex and the vagal response that normally produce tachycardia during nociception.¹⁸ Heart rate and blood pressure then become uncoupled, so the usual warning sign of light anesthesia or surgical stress is missing and a severe afterload surge can pass unnoticed. This is the “silent trap” for the anesthesia team. Open cholecystectomy is still needed when laparoscopy is

unsafe, and traction on the gallbladder and mesentery is a strong visceral stimulus.

Perioperative guidance concentrates on preoperative risk scores and says little about titration at the operating table, and descriptions of surge management usually assume invasive arterial monitoring. Many secondary hospitals cannot offer it, and radial cannulation carries its own risks in peripheral arterial disease and may delay urgent cases. Choices at emergence and in postoperative analgesia also matter, because coughing at extubation and breakthrough pain can each trigger a further afterload rise. A practical account of recognition and treatment with frequent non-invasive measurement is therefore useful.

This case report describes a 71-year-old man with advanced cardiometabolic multimorbidity who had an open cholecystectomy. It aims to show how hemodynamic uncoupling appeared during visceral traction and how it was recognized and treated, and to describe the associated plan for fluids, extubation and ward analgesia.

2. Methods

2.1. Report design and reporting guideline

This is a single case report. It follows the CARE guideline for case reports.

2.2. Setting

The patient was managed in the Department of Anesthesia, Sanjiwani Regional General Hospital, a regional general hospital in Gianyar, Bali, Indonesia. Care took place in the operating theater, the post-anesthesia care unit (PACU) and the surgical ward. Invasive arterial monitoring was not used because of limited institutional resources and severe peripheral arterial disease.

2.3. Patient information and case selection

The patient was a 71-year-old man with recurrent right upper quadrant colic, postprandial dyspepsia and vomiting. Magnetic resonance cholangiopancreatography showed symptomatic multiple cholelithiasis with chronic cholecystitis and extensive subhepatic adhesions. An open subcostal cholecystectomy was chosen because laparoscopic dissection was expected to be difficult and cardiopulmonary risk was high.

He had stage 2 hypertension at diagnosis, treated with ramipril 5 mg daily; coronary artery disease with exertional dyspnea of New York Heart Association (NYHA) class II, treated with bisoprolol 5 mg daily; and type 2 diabetes of more than 10 years' duration, treated with insulin glargine. Bilateral calf claudication indicated Fontaine stage II peripheral artery disease. Clopidogrel was stopped 5 days before surgery. Antihypertensive and insulin regimens continued through the perioperative period. Height was 172 cm, weight 80 kg and body mass index 27.04 kg/m². His American Society of Anesthesiologists (ASA) Physical Status was III and his Clinical Frailty Scale score was 3 ("Managing Well"). He could not climb two flights of stairs because of breathlessness, so functional capacity was below 4 metabolic equivalents (METs).

The case was selected because a marked rise in systolic pressure occurred without tachycardia in a patient with this combination of conditions, and because the surge was recognized and treated within minutes using non-invasive monitoring.

2.4. Case timeline

The course from the preoperative ward baseline through discharge on postoperative day (POD) 3 is shown in Table 1 using relative time points. Clock times are given for the intraoperative phase. The ward baseline is the 24-hour average before surgery and was used as the reference for percentage changes ($\Delta\%$), calculated as $[(T_n - T_0) / T_0] \times 100\%$.

2.5. Clinical findings and diagnostic assessment

Baseline blood pressure and heart rate were recorded over 24 hours on the ward to avoid "white-coat" reactivity in the holding area. Preoperative work-up included transthoracic echocardiography, a blood count and serum creatinine, glucose and electrolytes. The estimated glomerular filtration rate (eGFR) was calculated from creatinine with the CKD-EPI 2021 equation.

In the operating theater, blood pressure was measured with an automated oscillometric monitor and a cuff sized to arm circumference. Cycling was every 2 minutes in

stable phases and every minute at critical points: pre-oxygenation, induction, intubation, incision, peritoneal entry, deep gallbladder traction, cystic duct ligation, fascial closure and extubation. ECG leads II and V5 with automated ST-segment analysis, pulse oximetry, end-tidal CO₂, end-tidal sevoflurane and train-of-four (TOF) acceleromyography at the adductor pollicis were monitored continuously. Mean arterial pressure (MAP) was calculated as diastolic pressure + 1/3 (systolic – diastolic pressure), and the rate pressure product (RPP) as systolic pressure × heart rate. Reference thresholds were MAP below 65 mmHg for hypoperfusion, systolic pressure above 160 mmHg for a severe afterload surge, and RPP above 12,000 for myocardial oxygen demand stress. Left ventricular wall stress was approximated with the law of Laplace, $\sigma = (P \times r) / (2h)$.

Postoperatively, blood pressure, heart rate, respiratory rate, oxygen saturation, Richmond Agitation-Sedation Scale (RASS) score and pain on a 10-cm visual analog scale (VAS) at rest and on cough were recorded hourly. Venous blood was taken every 24 hours for hemoglobin, creatinine, glucose and electrolytes.

2.6. Therapeutic intervention

Co-induction followed a titrated sequence after 3 minutes of pre-oxygenation with 100% oxygen: midazolam 1 mg, fentanyl 2 mcg/kg (160 mcg) intravenously, then propofol 1.5 mg/kg (120 mg) injected slowly over 60 seconds, and atracurium 0.5 mg/kg (40 mg). Intubation used a GlideScope video laryngoscope with a size 4 blade and an 8.0-mm cuffed tube. Ventilation was volume-controlled (FiO₂ 0.5, tidal volume 500 mL, which is 6.25 mL/kg actual and 7.4 mL/kg predicted body weight, PEEP 5 cmH₂O, 12 breaths/min) and anesthesia was maintained with sevoflurane at 1.8 to 2.0% end-tidal. Intraoperative crystalloid was limited to 500 mL of Ringer's lactate, and no urinary catheter was placed.

A prespecified response to a pressure surge was used: NIBP cycling was changed to 1 minute, end-tidal sevoflurane was increased from 1.8% to 3.5%, and a rescue bolus of fentanyl 50 mcg was given intravenously.

Extubation was performed under sevoflurane (end-tidal about 1.2%) only if five criteria were met: (1) fasting of more than 8 hours with no gastroesophageal reflux; (2) TOF ratio above 0.9 after reversal with neostigmine 2.5 mg and glycopyrrolate 0.5 mg; (3) regular spontaneous breathing with tidal volume above 6 mL/kg and rate above 12 breaths/min; (4) central, conjugate pupils and absent swallowing; and (5) oropharyngeal suctioning under direct vision.

On the ward, fentanyl was infused at 12 mcg/hr by a dedicated electronic pump with four safety measures: a bed opposite the nursing station; hourly vital signs, manual respiratory rate and RASS for 6 hours, then every 2 hours; stopping of the infusion and emergency review for a respiratory rate below 10 breaths/min or RASS below –2; and naloxone 0.4 mg drawn up at the bedside. Oral paracetamol 1,000 mg every 8 hours was started when the infusion was stopped.

2.7. Outcome measures and follow-up

The main intraoperative outcome was the time needed to bring systolic pressure below 150 mmHg after the surge. Other outcomes were ECG ST-segment changes, creatinine and eGFR, glucose and potassium, hemoglobin, pain scores, respiratory rate, oxygen saturation, sedation score, wound

status and adverse events. The patient was followed until discharge on POD 3.

2.8. Ethics and consent

The report was cleared by the CMHC Research Center Ethics Committee, Indonesia (Protocol Approval Number: 2026/0418) and followed the Declaration of Helsinki. Written informed consent for care, for data use and for publication of the case, including figures and laboratory data, was obtained from the patient and his legal surrogate. Identifiers were removed.

3. Results

3.1. Presentation and baseline findings

The clinical course is summarized in Table 1 and the baseline findings in Table 2. On the ward, resting blood pressure averaged 138/84 mmHg (MAP 102.0 mmHg) and heart rate 72 bpm (RPP 9,936 mmHg·bpm). Hemoglobin was 13.5 g/dL, creatinine 1.10 mg/dL (eGFR 71.8 mL/min/1.73 m², CKD stage 2), random glucose 147 mg/dL and potassium 4.2 mmol/L. Echocardiography showed a left ventricular ejection fraction of 70.7% (Simpson biplane), concentric left ventricular hypertrophy (septal thickness 13.5 mm, relative wall thickness above 0.42) and grade I diastolic dysfunction (E/A below 0.8, deceleration time above 240 ms).

Table 1. Clinical timeline of the case from the preoperative ward baseline to discharge. Clock times are given for the intraoperative phase.

Time point	Clinical event	Key findings	Intervention
Ward baseline	Hospital ward, 24 hours before surgery	138/84 mmHg; MAP 102.0; HR 72; RPP 9,936	Ramipril, bisoprolol and insulin glargine continued
T0: 12:45 (operating theater entry)	Baseline surveillance in operating room	138/84 mmHg; MAP 102.0; HR 72; RPP 9,936	Pre-oxygenation with 100% O ₂ ; NIBP cycling set to 2-minute surveillance interval
T1: 12:55 (Induction & Intubation)	Pharmacological co-induction & video laryngoscopy	110/70 mmHg; MAP 83.3; HR 78; RPP 8,580; Δ% SBP/MAP/HR -20.3/-18.3/+8.3	Midazolam 1 mg, Fentanyl 160 mcg (2 mcg/kg), Propofol 120 mg (1.5 mg/kg), Atracurium 0.5 mg/kg; GlideScope intubation
T2: 13:00 (Surgical Incision)	Right subcostal (Kocher) abdominal incision	128/78 mmHg; MAP 94.7; HR 76; RPP 9,728; Δ% SBP/MAP/HR -7.2/-7.2/+5.6	Sevoflurane titrated to 1.8 – 2.0% in O ₂ /Air mixture; Volume Control Ventilation with PEEP 5 cmH ₂ O
T3: 13:10 (Abdominal Exploration)	Peritoneal entry & falciform ligament exposure	132/80 mmHg; MAP 97.3; HR 76; RPP 10,032; Δ% SBP/MAP/HR -4.3/-4.6/+5.6	Hemodynamic stability; depth of anesthesia stable; NIBP cycling maintained every 2 minutes
T4: 13:20 (Subhepatic Dissection)	Hepatic retraction & omental adhesion release	136/82 mmHg; MAP 100.0; HR 78; RPP 10,608; Δ% SBP/MAP/HR -1.4/-2.0/+8.3	Stable operative conditions; restrictive fluid infusion rate calibrated to 500 mL total volume
T5: 13:30 (Peak Mesenteric Traction)	Traction on gallbladder mesentery & Calot triangle	171/95 mmHg; MAP 120.3; HR 83; RPP 14,193; Δ% SBP/MAP/HR +23.9/+17.9/+15.3	Acute sympathetic surge triggered by visceral afferents; NIBP switched to 1-minute cycling; absence of tachycardia (HR 83 bpm)
T6: 13:32 (Pharmacological Intervention)	Immediate sympathetic attenuation protocol	162/90 mmHg; MAP 114.0; HR 82; RPP 13,284; Δ% SBP/MAP/HR +17.4/+11.8/+13.9	Sevoflurane deepened rapidly to 3.5%; intravenous rescue Fentanyl bolus (50 mcg) administered
T7: 13:34 (4-Minute Attenuation Endpoint)	Successful resolution of hypertensive surge	148/80 mmHg; MAP 102.7; HR 80; RPP 11,840; Δ% SBP/MAP/HR +7.2/+0.7/+11.1	Systolic BP successfully reduced to < 150 mmHg within 4 minutes; ECG Leads II & V5 show no ST depression
T8: 13:45 (Cystic Artery / Duct Ligation)	Cholecystectomy completion & bed hemostasis	134/78 mmHg; MAP 96.7; HR 76; RPP 10,184; Δ% SBP/MAP/HR -2.9/-5.2/+5.6	Sevoflurane returned to maintenance 1.8%; complete abdominal hemostasis verified; warm saline irrigation
T9: 13:55 (Abdominal Wall Closure)	Fascial closure & criterion-based extubation prep	132/76 mmHg; MAP 94.7; HR 75; RPP 9,900; Δ% SBP/MAP/HR -4.3/-7.2/+4.2	Neuromuscular blockade reversal; TOF ratio > 0.9 confirmed; spontaneous Vt > 6 mL/kg, RR > 12 bpm
T10: 14:00 (Deep Extubation)	Tracheal extubation under surgical anesthetic plane	136/78 mmHg; MAP 97.3; HR 74; RPP 10,064; Δ% SBP/MAP/HR -1.4/-4.6/+2.8	Smooth deep extubation avoiding coughing, bucking, and emergence hypertensive afterload spikes (> 200 mmHg)
T11: 14:15 (PACU Stabilization)	Post-anesthesia care unit initial monitoring	130/80 mmHg; MAP 96.7; HR 74; RPP 9,620; Δ% SBP/MAP/HR -5.8/-5.2/+2.8	Awake, calm, oriented; pain score VAS 1/10; initiated continuous ward-based low-dose fentanyl infusion at 12 mcg/hr
POD 1 to POD 3	Ward recovery	Creatinine 1.05, 1.02, 1.00 mg/dL; VAS at rest 1–2, 1, 0–1; no respiratory depression	Fentanyl 12 mcg/hr until morning of POD 2; oral paracetamol; discharge on POD 3

Notes: BP, blood pressure (SBP/DBP, mmHg); MAP, mean arterial pressure (mmHg); HR, heart rate (bpm); RPP, rate pressure product (SBP × HR, mmHg·bpm); Δ%, percentage change from the ward baseline; NIBP, non-invasive blood pressure; PACU, post-anesthesia care unit; POD, postoperative day; TOF, train-of-four; VAS, visual analog scale. T0 shows the ward baseline average; T5 and T6 times are as submitted.

Table 2. Baseline demographic, anthropometric, cardiometabolic and preoperative findings.

Parameter	Observed value	Reference or classification
Demographic Status	71 years / Male	Geriatric population (≥ 65 years)
Surgical Indication	Symptomatic Multiple Cholelithiasis	Open Cholecystectomy Indication
Anthropometry: Height & Weight	172 cm / 80 kg	Standard Adult Population
Body Mass Index (BMI)	27.04 kg/m ²	Overweight (25.0 – 29.9 kg/m ²)
ASA Physical Status	Class III	Severe systemic disease
Clinical Frailty Scale (CFS)	Score 3 (Managing Well)	Scale 1 – 9 (1: Very Fit, 9: Terminally Ill)
Cardiovascular Pathology	Stage II Hypertension & CAD	AHA/ACC Stage II ($\geq 140/90$ mmHg)
Heart Failure Symptomatology	NYHA Class II Exertional Dyspnea	New York Heart Association Class I–IV
Endocrine Comorbidity	Type II Diabetes Mellitus (> 10 years)	Insulin-dependent T2DM
Vascular History	Intermittent Claudication	Fontaine Stage II Peripheral Artery Disease
Functional Reserve	< 4 Metabolic Equivalents (METs)	Threshold for Major Surgery: ≥ 4 METs
24-Hour Ward Baseline Hemodynamics	138/84 mmHg, HR 72 bpm	Target Mean Arterial Pressure (MAP): 102 mmHg
Pharmacotherapy: Antiplatelet	Clopidogrel (Stopped 5 days pre-op)	5-day therapeutic washout window
Pharmacotherapy: Antihypertensives	Ramipril 5 mg & Bisoprolol 5 mg	Maintained up to morning of surgery
Hematological Reserve	Hemoglobin 13.5 g/dL	13.0 – 17.0 g/dL
Renal Filtration Reserve	Creatinine 1.10 mg/dL, eGFR 71.8 mL/min/1.73 m ²	eGFR 60–89 mL/min/1.73 m ² (CKD stage 2)
Glycemic & Electrolyte Status	Random Glucose 147 mg/dL, K ⁺ 4.2 mmol/L	Glucose 70–140 mg/dL, K ⁺ 3.5–5.0 mmol/L
Echocardiographic Systolic Function	LVEF 70.7% (Simpson biplane)	Normal Systolic Reserve: $\geq 50\%$
Echocardiographic Diastolic Function	Grade I Diastolic Dysfunction (E/A < 0.8)	Normal Diastolic Filling: E/A > 1.0
Left Ventricular Structural Geometry	Concentric Left Ventricular Hypertrophy	Relative Wall Thickness > 0.42

Notes: ACC, American College of Cardiology; AHA, American Heart Association; ASA, American Society of Anesthesiologists; BMI, body mass index; CAD, coronary artery disease; CFS, Clinical Frailty Scale; CKD, chronic kidney disease; E/A, ratio of early to late transmitral filling velocities; eGFR, estimated glomerular filtration rate (recalculated with CKD-EPI 2021); LVEF, left ventricular ejection fraction; METs, metabolic equivalents; NYHA, New York Heart Association; PAD, peripheral artery disease; T2DM, type 2 diabetes mellitus.

3.2. Diagnostic findings

Induction at 12:55 (T1) was followed by a fall in blood pressure to 110/70 mmHg (MAP 83.3 mmHg, heart rate 78 bpm, RPP 8,580 mmHg·bpm; $\Delta\%$ SBP -20.3% , $\Delta\%$ MAP -18.3% , $\Delta\%$ HR $+8.3\%$). Intubation was achieved on the

first attempt without airway trauma or desaturation. After the right subcostal (Kocher) incision at 13:00 (T2), exploration (T3) and subhepatic mobilization (T4), pressures were stable (128/78 to 136/82 mmHg; MAP 94.7 to 100.0 mmHg; heart rate 76 to 78 bpm; Figures 1 and 2).

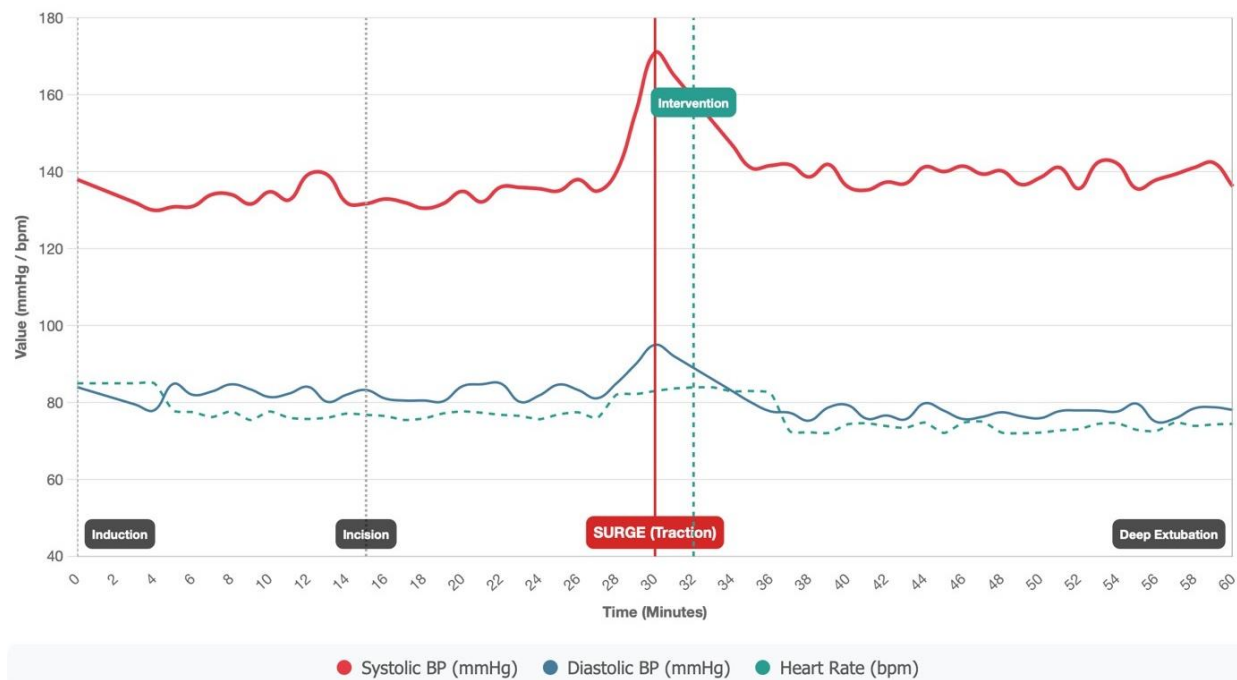


Figure 1. Intraoperative hemodynamic trend over 60 minutes of open cholecystectomy: baseline stability, systolic pressure of 171 mmHg at minute 30 and stabilization after the intervention. SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate.

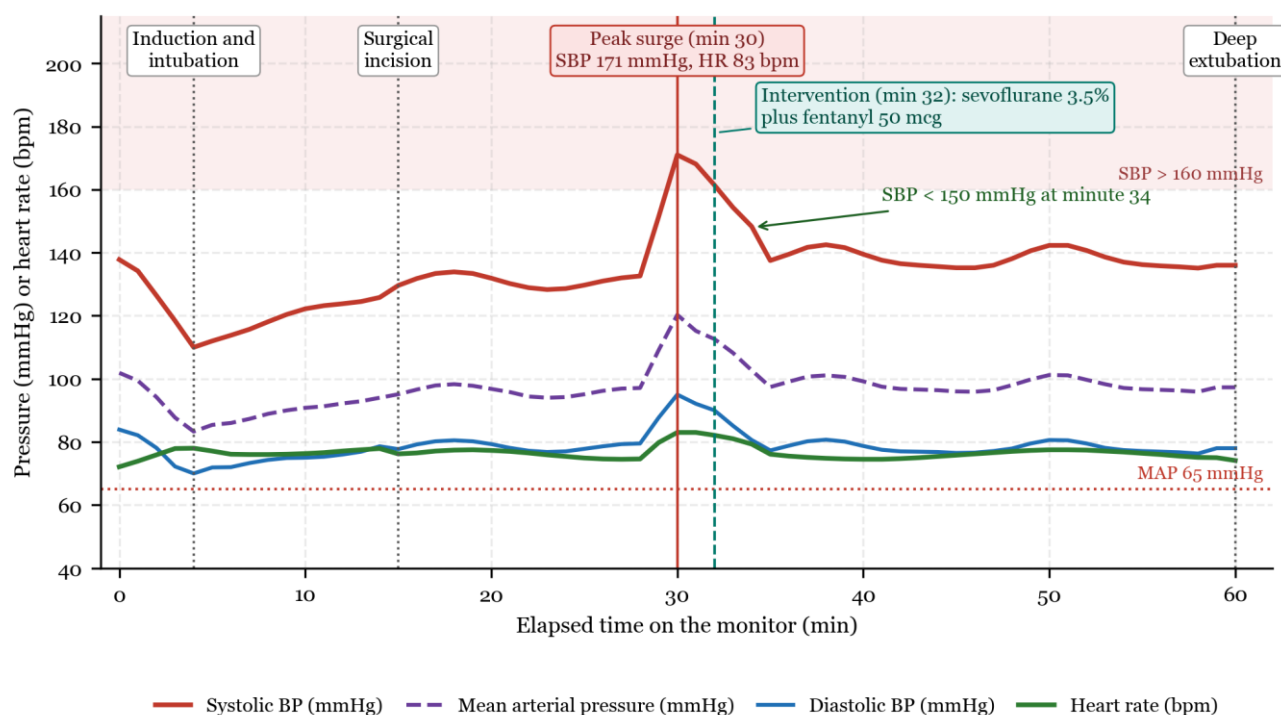


Figure 2. Hemodynamic trajectory and surgical milestones: systolic, diastolic and mean arterial pressure and heart rate. The surge at minute 30 (SBP 171 mmHg, HR 83 bpm) and systolic pressure below 150 mmHg at minute 34 are marked. Values between recorded oscillometric readings are interpolated, as in the submitted figure.

At 13:30 (T5), during deep dissection of the triangle of Calot with traction on the gallbladder mesentery, blood pressure rose to 171/95 mmHg (MAP 120.3 mmHg). Systolic pressure was 23.9% and MAP 17.9% above the ward baseline, and systolic pressure was 55.5% above the

post-induction value. Heart rate was 83 bpm (+15.3%), and RPP rose by 42.8% to 14,193 mmHg·bpm, above the 12,000 threshold (Figure 3A). The ECG showed no ST-segment depression in leads II and V5. The wall stress index was modeled at 145.3 kPa (Figure 3B).

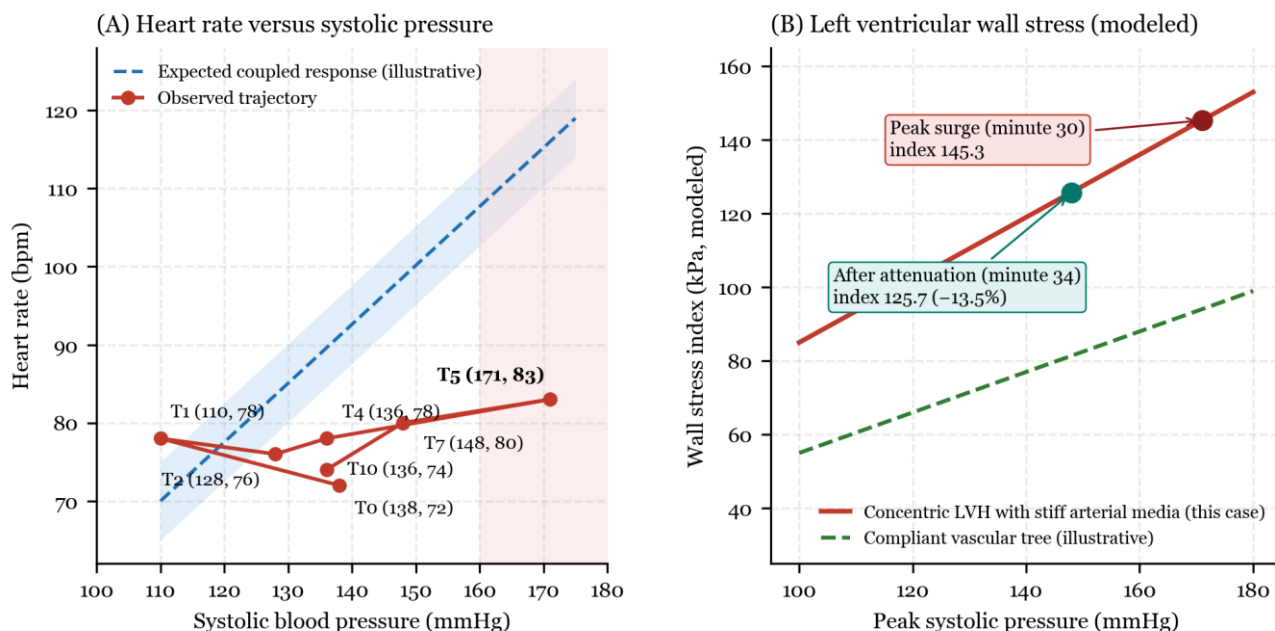


Figure 3. (A) Heart rate versus systolic pressure at the main time points; the shaded diagonal band shows an illustrative coupled response. (B) Modeled left ventricular wall stress index versus peak systolic pressure for concentric hypertrophy with stiff arterial media and for a compliant vascular tree (illustrative). T0 to T10 are the time points of Table 1.

3.3. Treatment course and outcome

Sevoflurane was increased to 3.5% and fentanyl 50 mcg was given at about 13:32 (T6). At 13:32 systolic pressure

was 162 mmHg (MAP 114.0 mmHg, heart rate 82 bpm), and at 13:34 (T7) it was 148/80 mmHg (MAP 102.7 mmHg, heart rate 80 bpm). That is 4 minutes after the surge began and 2 minutes after the interventions. After ligation of the

cystic duct and artery (T8, 13:45) sevoflurane was returned to 1.8% and pressure was 134/78 mmHg. The gallbladder was removed intact, and blood loss was estimated at less than 50 mL.

Creatinine was 1.10 mg/dL before surgery and 1.05, 1.02 and 1.00 mg/dL on POD 1, 2 and 3 (eGFR 71.8, 75.9, 78.6

and 80.5 mL/min/1.73 m²; Table 3 and Figure 4A). MAP stayed above 83 mmHg during surgery. Urine output on POD 1 was 0.78 mL/kg/h without diuretics. Glucose rose to 158 mg/dL at the surge and was 142, 134 and 126 mg/dL on POD 1, 2 and 3. Potassium stayed between 4.0 and 4.2 mmol/L, and hemoglobin was 12.9 g/dL on POD 1 and 13.0 g/dL on POD 3 (Table 3, Figure 4B).

Table 3. Serial laboratory, hemodynamic, fluid and analgesia values across preoperative, intraoperative and postoperative milestones.

Parameter	Reference	Preop	Intraop (T5)	POD 1	POD 2	POD 3
Hemoglobin (g/dL)	13.0 – 17.0	13.5	-	12.9	12.8	13.0
Hematocrit (%)	40.0 – 50.0	41.2	-	39.4	39.1	39.8
Serum Creatinine (mg/dL)	0.70 – 1.20	1.10	-	1.05	1.02	1.00
eGFR (mL/min/1.73 m ² , CKD-EPI 2021)	≥ 60.0 (Stage 2: 60-89)	71.8	-	75.9	78.6	80.5
Random Blood Glucose (mg/dL)	70–140	147	158	142	134	126
Serum Potassium (mmol/L)	3.5 – 5.0	4.2	-	4.0	4.1	4.2
Serum Sodium (mmol/L)	135 – 145	139	-	140	138	139
Total Fluid Input (mL)	Variable	0	500 (RL)	1,500 (Total/24h)	1,800 (Oral+IV)	Oral Only
Urine Output (mL/kg/h)	≥ 0.50	Pre-op Void	-	0.78	0.85	0.92
VAS at rest (0–10)	0–10	0 / 10	Intraoperative	1 – 2 / 10	1 / 10	0 – 1 / 10
VAS on cough (0–10)	0–10	0 / 10	Intraoperative	3–4 / 10	2–3 / 10	2 / 10
Respiratory Rate (breaths/min)	12 – 20	16	12 (Ventilator)	14 – 16	14 – 16	16
Peripheral Oxygen Saturation (SpO ₂)	95 – 100%	98%	99% (FiO ₂ 0.5)	97 – 98%	98%	98%
RASS score	0	0	-5 (General Anesth)	0 to -1	0	0

Notes: Units are as given in the parameter column. eGFR was recalculated from creatinine, age and sex with the CKD-EPI 2021 equation. “-” means not measured. OIRD, opioid-induced respiratory depression; POD, postoperative day; RASS, Richmond Agitation-Sedation Scale; RL, Ringer’s lactate; SpO₂, peripheral oxygen saturation; VAS, visual analog scale.

All five extubation criteria were met (neostigmine and glycopyrrolate reversal; TOF ratio above 0.9; spontaneous tidal volume 520 mL). The patient was extubated at 14:00 (T10) without cough, bucking or breath-holding. Pressure was 136/78 mmHg (MAP 97.3 mmHg, heart rate 74 bpm, RPP 10,064 mmHg·bpm), with Δ% SBP -1.4%, Δ% MAP -4.6% and Δ% HR +2.8%. A nasopharyngeal airway and facemask oxygen at 5 L/min kept SpO₂ at 99%. At 14:15 (T11) in the PACU he was awake, oriented and calm, with blood pressure 130/80 mmHg and heart rate 74 bpm, and without delirium, shivering or respiratory compromise.

The fentanyl infusion at 12 mcg/hr started on ward arrival. Resting pain was VAS 1 to 2 of 10 on POD 1, 1 on POD 2 and 0 to 1 on POD 3. Pain on cough was VAS 3 to 4 on POD 1, 2 to 3 on POD 2 and 2 on POD 3. Respiratory rate was 14 to 16 breaths/min, SpO₂ was 97 to 98% on room air, and RASS was 0 to -1. There was no bradypnea, desaturation or oversedation. The infusion was stopped on the morning of POD 2. The patient mobilized on POD 1, resumed oral feeding on POD 1, and passed flatus on POD 2 (Figure 4). The wound stayed clean and dry, and he was discharged on POD 3 without cardiovascular, pulmonary or renal adverse events (Figure 4D).

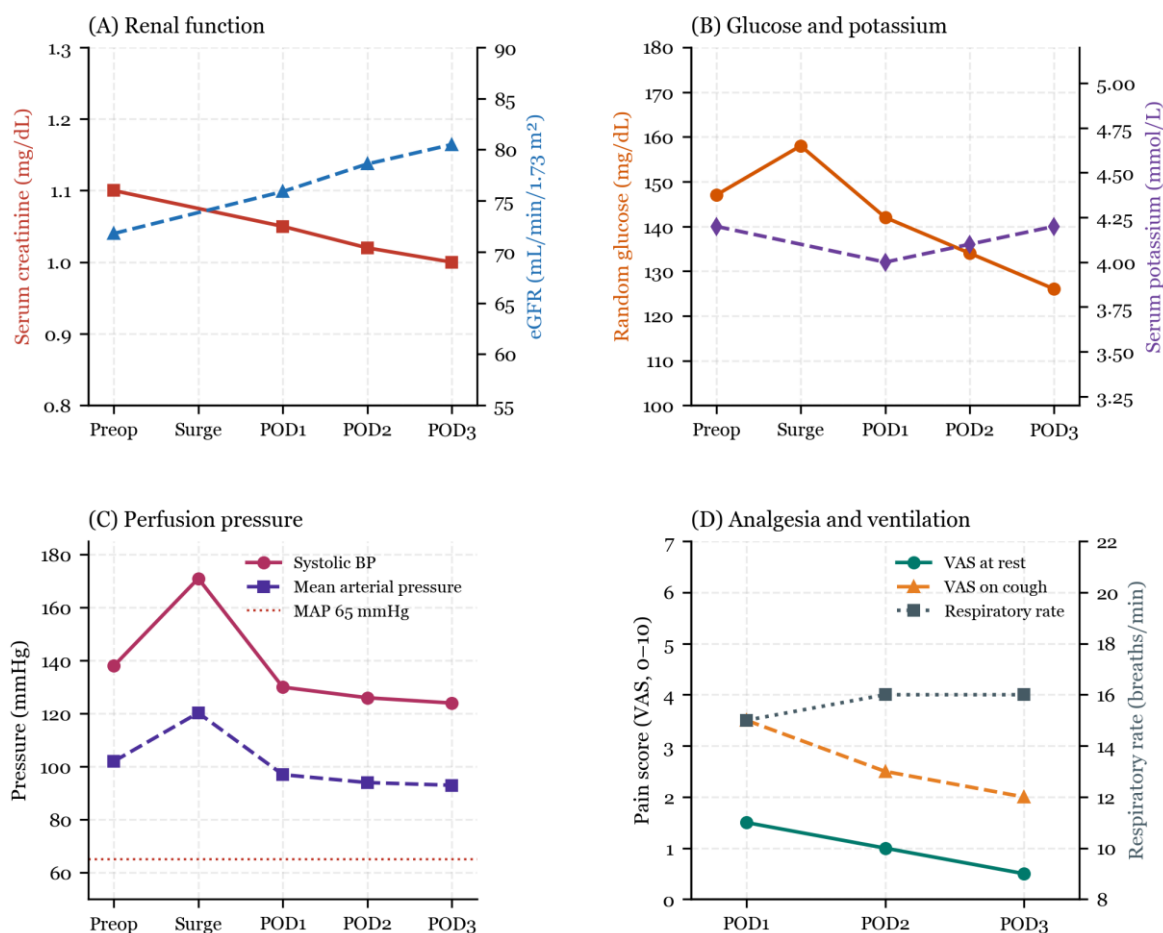


Figure 4. Perioperative trajectories. (A) Serum creatinine and eGFR. (B) Random glucose and serum potassium. (C) Systolic and mean arterial pressure. (D) Pain scores at rest and on cough, and respiratory rate. POD, postoperative day; VAS, visual analog scale.

4. Discussion

4.1. Principal lesson of the case

In an older diabetic patient treated with a beta-blocker, a sudden rise in systolic pressure with almost no rise in heart rate was the only sign of a visceral nociceptive surge. It was recognized because pressure was measured every minute, and it responded within minutes to deeper sevoflurane and a small fentanyl bolus.

4.2. Comparison with the published literature

Cardiac autonomic neuropathy in older diabetic surgical patients has been linked to hemodynamic instability. In a study of older patients under general anesthesia, Siripruekpong et al. found that co-occurring diabetes and cardiac autonomic neuropathy influenced post-induction hypotension.¹⁹ Our patient had the opposite problem, a pressor surge during surgical traction, which suggests that the same neuropathy can remove protective chronotropic feedback in either direction. Slow induction in older patients has been proposed to prevent burst suppression on processed EEG,²⁰ which is consistent with the 60-second propofol injection used here. For fluids, the RELIEF trial compared restrictive and liberal regimens in major abdominal surgery and reported more acute kidney injury with the restrictive regimen.²⁰ Our restriction to 500 mL was therefore not supported by that trial, and the stable creatinine we observed is a single observation.

4.3. Pathophysiological or mechanistic interpretation

What was observed is a rise in systolic pressure to 171 mmHg with a heart rate of 83 bpm, its treatment, and an uneventful course. The mechanism below is inferred. Chronic hyperglycemia and age are thought to stiffen the arterial media through glycation and calcification, which reduces Windkessel buffering and fixes vascular resistance,^{8–10} and arterial stiffness has been associated with cardiac autonomic neuropathy in diabetes.^{11–17} A catecholamine burst from visceral afferents would then be converted into a large pressure rise without the usual rise in heart rate, an effect that bisoprolol would add to. Autonomic function tests were not done, so neuropathy is assumed and not demonstrated, and arterial stiffness was not measured.

Higher systolic pressure raises left ventricular wall stress and oxygen demand, as the law of Laplace describes. Figure 3B shows a modeled index, not a measurement. Deeper sevoflurane relaxes vascular smooth muscle and lowers sympathetic outflow, and fentanyl reduces visceral afferent input, so the two may act together. A concurrent contribution from light anesthesia, hypercarbia or a different mechanism, such as the pattern described for mesenteric traction syndrome, cannot be excluded.

For postoperative analgesia, intermittent boluses produce peaks and troughs of plasma concentration, whereas a continuous infusion can keep concentration within a narrow window (Figure 5; illustrative model curves, not measured concentrations). Figure 6 summarizes the

proposed sequence from the substrate to the management steps.

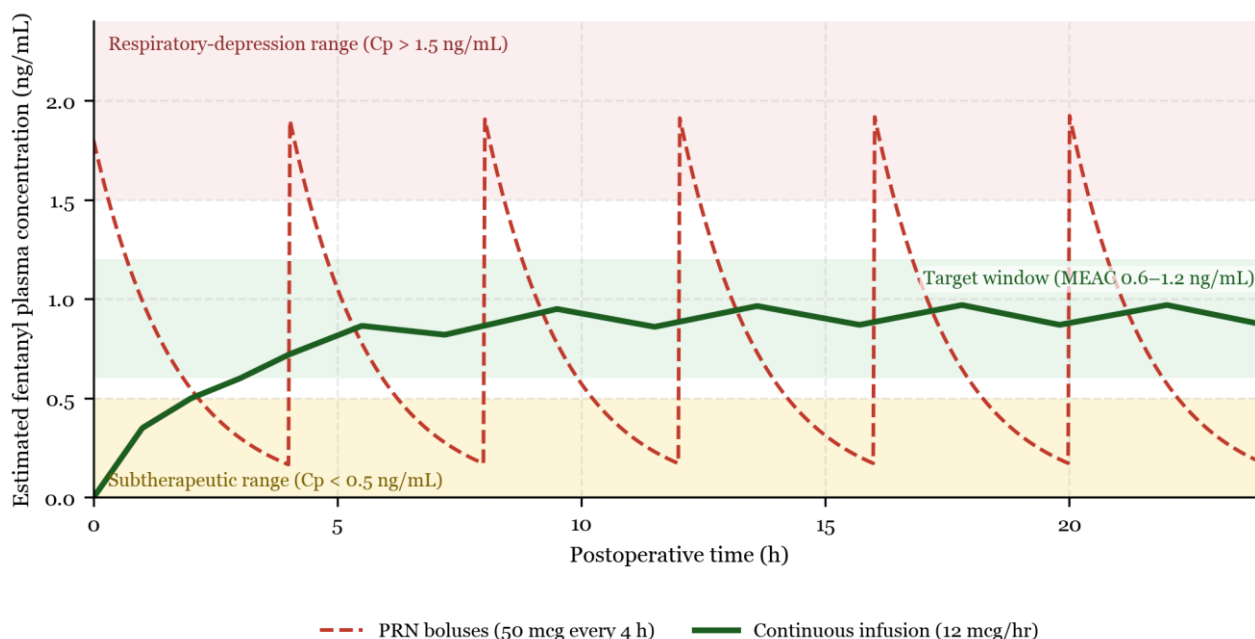


Figure 5. Illustrative model curves of estimated fentanyl plasma concentration for a continuous infusion of 12 mcg/hr and for intermittent boluses of 50 mcg every 4 hours. Concentrations were not measured. MEAC, minimum effective analgesic concentration; PRN, as needed.

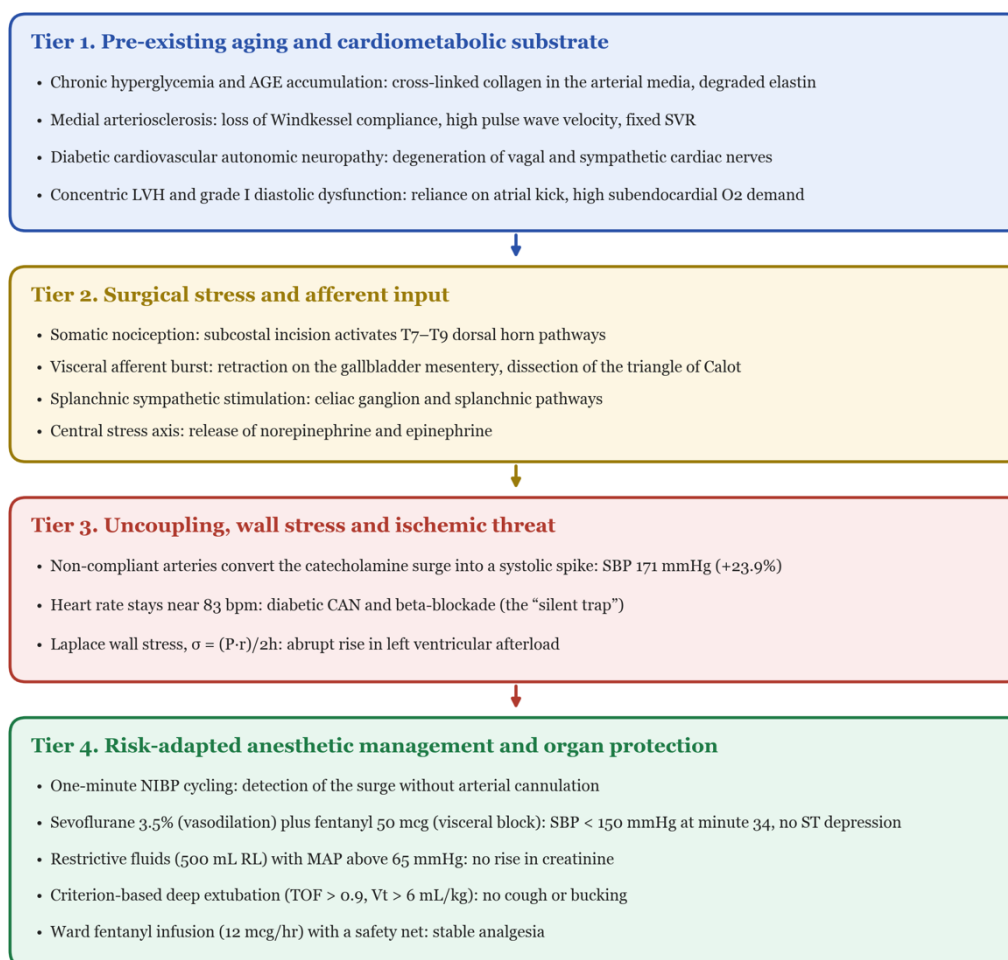


Figure 6. Proposed sequence from the pre-existing substrate through surgical stimulation and hemodynamic uncoupling to risk-adapted management. AGE, advanced glycation end-products; CAN, cardiovascular autonomic neuropathy; LVH, left ventricular hypertrophy; NIBP, non-invasive blood pressure; SVR, systemic vascular resistance; TOF, train-of-four; Vt, tidal volume.

4.4. Implications for practice

In older diabetic or beta-blocked patients, a rise in blood pressure without tachycardia during visceral traction should be treated as nociception until proven otherwise. Measuring pressure every minute at stimulating steps is feasible without an arterial line, and a pre-agreed response (deeper volatile anesthetic plus a small opioid bolus) shortens the time to control. Deep extubation against written criteria was uneventful here, but diabetic autonomic neuropathy can delay gastric emptying, so the aspiration risk needs separate consideration. Continuous opioid infusion on a general ward needs a defined monitoring plan. Transferability is limited to similar patients and to units that can provide this level of nursing surveillance.

4.5. Strengths

The record is complete and timed, derived values reproduce from the measured ones, the surge and its treatment are documented to the minute, the extubation and ward measures are written as explicit criteria, and consent and ethics approval are documented.

4.6. Limitations

This is a single case and cannot establish causation, prevalence or effect. Arterial waveform monitoring, stroke volume variation and processed EEG were not used, and short pressure swings between cuff cycles could have been missed. Autonomic function and arterial stiffness were not measured. Cardiac biomarkers (high-sensitivity troponin, NT-proBNP) were not measured after surgery, so myocardial injury after non-cardiac surgery cannot be excluded, and the absence of ECG changes does not prove myocardial protection. Postoperative pressures are not reported in the text. The wall stress index and the pharmacokinetic curves are modeled.

4.7. Recommendations for future work

Prospective multicenter registries of older vasculopathies undergoing major abdominal surgery, with autonomic function testing, arterial stiffness measurement and postoperative troponin, would be needed to test whether pressure-led titration with non-invasive monitoring reduces myocardial injury.

5. Conclusion

This case shows that, in an older diabetic patient on a beta-blocker, a rise in systolic pressure without tachycardia may be the only sign of visceral nociception during open cholecystectomy. One-minute non-invasive pressure measurement, deeper sevoflurane and a small fentanyl bolus controlled the surge within minutes without ECG changes, and the patient recovered without cardiovascular, pulmonary or renal events. The report claims no more than one case supports: absence of tachycardia should not be read as adequate anesthetic depth in similar patients, and the approach needs testing in larger prospective series.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under reference number 2026/0418 issued in 2026. The study followed the Declaration of Helsinki. Written informed consent was obtained from the patient.

Consent for publication. Written informed consent for publication of this case and any accompanying images and data was obtained from the patient and his legal surrogate.

Availability of data and materials. The datasets generated and analyzed during this case report are available from the corresponding author on reasonable request.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. L.A.M.: Conceptualization; Methodology; Investigation; Data curation; Formal analysis; Visualization; Writing – original draft. K.J.U.D.: Methodology; Validation; Supervision; Writing – review & editing. Both authors read and approved the final manuscript.

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Conflict of interest statement. The authors declare that they have no competing interests.

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